

***PRE-FILED TESTIMONY
OF DAVID O. CARPENTER, M.D.
MPUC Docket No. 2011-00262***

1 **Q. Please state your name and business address.**

2 A. My name is David O. Carpenter. My business address is:

3 Institute for Health and the Environment
4 University at Albany
5 Five University Place, Room A217
6 Rensselaer, NY 12144-3456

7 **Q. Briefly state your occupation, educational background and current**
8 **employment.**

9 A. I am a public health physician and professor, with a medical degree from Harvard
10 Medical School. I have held various positions in the public health field. My
11 current title is Director of the Institute for Health and the Environment at the
12 University at Albany and Professor of Environmental Health Sciences within the
13 School of Public Health. In addition I am an Honorary Professor, Queensland
14 Children's Medical Research Unit, University of Queensland, Brisbane, Australia.

15 Formerly, I was the Director of the Wadsworth Center for Laboratories and
16 Research of the New York State Department of Health and the Dean of the School
17 of Public Health at the University of Albany, while remaining employed by the
18 New York State Department of Health. I assumed my current position in 1998.

19 I served as the Executive Secretary to the New York State Powerlines
20 Project in the 1980s, a program of research that showed that children living in
21 homes with elevated magnetic fields coming from powerlines suffered from an

1 elevated risk of developing leukemia, and that electromagnetic field (EMF)
2 exposure altered a variety of responses studied in animals and in cellular systems.
3 After this, I became the spokesperson on EMF issues for New York during the
4 time of my employment in the Department of Health.

5 Attached as Exhibit A is my *curriculum vitae*.

6 **Q. Are you a member of any professional organizations?**

7 A. I participate in many international, national, state and local organizations and
8 committees as listed in my *curriculum vitae* along with the Honors, Awards, and
9 Fellowships I have received.

10 **Q. Have you authored any papers or journal articles?**

11 A. I have authored over 350 major publications in peer-reviewed scientific journals,
12 have edited five books and have numerous other publications as listed in my
13 *curriculum vitae*.

14 **Q. Briefly describe your work and experience related to the study of health risks**
15 **related to electromagnetic fields and radio frequency waves in the 30 MHz to**
16 **300 GHz range ("RF"). Identify any studies or published writings on the**
17 **subject.**

18 I have published several reviews and have edited two books on the Biologic
19 Effects of Electric and Magnetic Fields. I am also a Co-Editor and a Contributing
20 Author of the *BioInitiative Report: A Rationale for a Biologically-based Public*
21 *Exposure Standard for Electromagnetic Fields (ELF and RF)*
22 www.bioinitiative.org. This report was first published in 2007, and has just now

1 been updated in 2012. The *BioInitiative Report* documents bioeffects, adverse
2 health effects and public health conclusions about impacts of electromagnetic
3 radiation (electromagnetic fields including extremely-low frequency ELF-EMF
4 and radiofrequency /microwave or RF-EMF fields). I will refer to specific
5 sections of the report where appropriate but I also reference the entire report as a
6 comprehensive and up-to-date review of the scientific information on this subject.

7
8 In 2009, I was invited to present to the President's Cancer Panel on the
9 subject of power line and radiofrequency fields and cancer, and have also testified
10 on this issue before the United States House of Representatives.

11 **Q. Are you familiar with peer-reviewed studies addressing the biological effects**
12 **of exposure to low-level RF, and their potential health effects?**

13 A. There are many peer-reviewed studies reporting biological effects and health risks
14 related to low-level RF exposure. A comprehensive listing of these publications is
15 found in the *Bioinitiative Report*, which includes both positive and negative
16 research studies. In this testimony, I will not list peer-reviewed publications dated
17 prior to 2000 or any covered by publications that are systematic reviews or meta-
18 analyses reported after that time. I will focus on human studies, and only cover
19 briefly the huge number of cellular and animal studies. In my judgment the
20 scientific results of greatest importance, consistency and relevance to human
21 health are listed first.

1 Q. Is there reliable evidence from epidemiological studies to support the conclusion
2 that low-level RF (below the level at which thermal effects are confirmed) can
3 cause adverse health effects?

4 There is consistent evidence for harm from low-level RF radiation in
5 studies of individuals using cell phones for prolonged periods of time, which gives
6 a localized exposure to the ipsilateral brain, auditory nerve and parotid gland in the
7 cheek. There have been seven major publications that are either meta-analyses or
8 pooled analyses that evaluate all of the earlier literature, and most find statistically
9 significant relations between elevated exposure to radiofrequency radiation from
10 cell phones and increased risk of brain cancer. I will also discuss several recent
11 individual studies on cell phone exposure and some relevant studies on radio
12 transmission exposure. I will refer frequently to the odds ratio (OR) or risk ratio
13 (RR). These are statistical analysis terms that are used to determine whether or
14 not results are statistically significant. The standard use is to give an OR or RR
15 followed by the 95% confidence interval. Thus, if there is no difference between
16 the "exposed" and "control" populations, the OR or RR will be 1. If there is an
17 elevated risk the OR or RR will be greater than 1.0, whereas if the exposure
18 reduces risk of disease the OR or RR will be less than 1.0. For exposures that
19 increase risk, results are considered to be statistically significant if the 95% CI has
20 a lower bound that is greater than 1, which is to say that there is less than a 5%
21 possibility that the result occurred by chance. The seven major meta-analysis and
22 pooled analysis publications I mentioned are summarized below:

1
2 a. Hardell L, Carlberg M, Soderqvist F, Mild KH. 2008. Meta-analysis
3 of long-term mobile phone use and the association with brain tumours.
4 *Internat J Oncology* 12: 1097-1103. In ten studies of glioma, cell phone
5 use for more than ten years gave an OR of 1.2 (95%CI=0.8-1.9) (thus this
6 result would not be considered to be significant, since the lower bound is
7 less than 1.0). For ipsilateral cell phone use for more than 10 year the OR =
8 2.0 (1.2-3.4) (thus this result is statistically significant, since the lower
9 bound is greater than 1.0). There was also a significant association for
10 acoustic neuroma and ipsilateral cell phone use for ten years or more, but
11 no relation for meningioma.

12
13 b. Kundi M. 2009. The controversy about a possible relationship
14 between mobile phone use and cancer. *Environ Health Perspect* 117: 316-
15 324. Reviewed data from 33 epidemiological studies and concludes that
16 the combined OR = 1.5 (1.2-1.8) for glioma and 1.1 (0.8-1.4) for
17 meningioma.

18
19 c. Myung SK, Ju W, McDonnell DD, Lee YJ, Ksazinet G, Cheng CT,
20 Moskowitz JM. 2009. Mobile phone use and risk of tumors: A meta-
21 analysis. *J Clin Oncol* 27:5565-5572. Reviewed 465 publications that
22 reported on 12,344 cases of cancer and 25,572 controls. Risk of developing
23 brain cancer was OR = 1.8 (1.04-1.34) for more than ten years use.

24
25 d. Ahlbom A, Feychting M, Green A, Kheifet L, Savitz DA and
26 Swedlow AJ (ICNIRP Standing Committee on Epidemiology). 2009.
27 Epidemiologic evidence on mobile phones and tumor risk: A review.
28 *Epidemiology* 20: 639-652. Comment that most studies of glioma show
29 small increased or decreased risk among users, although a subset of studies
30 show appreciably elevated risks. They then argue that there are
31 methodological reasons for these positive studies.

32
33 e. Khurana VG, Teo C, Kundi M, Hardell L and Carlberg. 2009. Cell
34 phones and brain tumors: a review including the long-term epidemiological
35 data. *Surg Neurol* 72: 205-214. Meta-analysis of 11 studies. They
36 conclude that using a cell phone for more than 10 years approximately
37 doubles the risk of being diagnosed with a brain tumor (glioma, OR = 1.9,
38 1.4-2.4, and acoustic neuroma, OR = 1.6, 1.1-2.4) on the ipsilateral side of
39 the head.

40
41 f. Repacholi MH, Lerchl A, Roosli M, Sienkiewica Z, Auvinen A, et
42 al. 2012. Systematic review of wireless phone use and brain cancer and
43 other head tumors. *Bioelectromagnetics* 33: 187-206. Meta-analysis of

studies shows no relationship between brain cancers and ever use of a mobile phone (for glioma, OR = 1.07, 0.89-1.29, based on eight studies and use for one to five years), but there is sparse data on long-term use. Meta-analysis of oncogenicity, tumor promotion and genotoxicity studies also showed no statistically significant relationship between RF exposure and genotoxic damage to brain cells.

g. Hardell L, Carlberg M, Hansson Mild K. 2012. Use of mobile phones and cordless phones is associated with increased risk for glioma and acoustic neuroma. *Pathophysiology* doi:10.1016/j.pathophys.2012.11.001. In a review of current evidence they report that a meta-analysis for glioma in the temporal lobe, gave an OR = 1.74 (1.04-2.81). For ipsilateral mobile phone use for 1640 hours or more gave an OR = 2.29 (1.56-3.37). For acoustic neuroma, use for more than 10 years gave an OR = 1.81 (0.73-4.45), and for ipsilateral cumulative use of the same duration the OR = 2.55 (1.50-4.40).

A partial list of recent research studies on cell phone exposure (not reviews) are listed below:

a. The INTERPHONE Study Group. 2010. Brain tumour risk in relation to mobile telephone use: results of the INTERPHONE international case-control study. *Int J Epidemiol* 39:675-694. While ever vs. never using a cell phone did not increase risk of brain cancer, there was a significant OR= 2.18 (1.43-3.31) for use for ten or more years, OR=1.82 (1.15-2.89) for use for 1640 hours or more and OR=1.31 (0.82-2.11) for more than 270 calls, all for glioma. No significant relations were seen for meningioma. It should be noted that separate INTERPHONE results have been published for Sweden (Lonn et al. 2005. *J Epidemiol* 161: 526-636) and Germany (Schüz et al. 2006. *J Epidemiol* 163: 512-520). The German, but not the Swedish study, reported elevated rates of glioma with cell phone use for more than 10 years.

b. The INTERPHONE Study Group. 2011. Acoustic neuroma risk in relation to mobile telephone use: Results of the INTERPHONE international case-control study. *Cancer Epidemiol* 35: 453-464. Ever using a cell phone was not associated with elevated risk, nor was use for 10 years or more. For more than 1640 hours of use the OR was 2.79 (1.51-5.16).

c. Larjavaara S, Schüz J, Swerdlow A, Feychting M, Johansen C, et al. 2011. Location of gliomas in relation to mobile telephone use: A case-case and case-specular analysis. *Am J Epidemiol* 174: 2-11. Investigated 888

gliomas from seven European countries (INTERPHONE data) to determine whether the gliomas were located on the side of the head where the cell phone was regularly used. They found an elevated, but not significant, relationship in case-case analysis, but no difference in the case-specular analysis.

d. Levis AG, Minicuci N, Ricci P, Gennaro V, Gabisa S. 2011. Mobile phones and head tumours. The discrepancies in cause-effect relationships in the epidemiological studies – how do they arise? *Environ Health* 10:59 doi: 10.1186/1476-069X-10-59. When studies that were blinded, free from errors and bias were considered, cell phone use for more than ten years resulted in a near doubling in ipsilateral glioma and acoustic neuroma.

e. Aydin D, Feychting M, Schuz J, Tynes T, Andersen TV, et al. 2011. Mobile phone use and brain tumors in children and adolescents: A multicenter case-control study. *J Natl Cancer Inst* 103: 1264-1276. Studied all children between ages 7-19 with a brain tumor in four European countries. OR for regular mobile phone users was 1.36 (0.92-2.02), and for those using phones at least five years was 1.26 (0.70-2.28). Thus, rates were elevated but not statistically significant and there was no evidence of a dose-response relationship. However, for more than 2.8 years subscription the OR = 2.15 (1.07-4.29), and almost all ORs were elevated when comparing users to non-users. There were highly significant ORs with time since first use, cumulative duration of subscriptions, cumulative duration of call and cumulative number of calls, and these were found on both ipsi- and contralateral sides of the head. This is important, since the evidence for elevated risk only ipsilateral comes from data only on adults, and other evidence indicates greater penetration into the brain of a child. None-the-less, the authors conclude that this study provides no support for a relationship between cell phone use and brain cancer in children and adolescents because of the failure to find a dose-response relationship. The conclusions drawn in this study have been questioned by Soderqvist et al. (*Environ Health* 2011. 10:106) on the basis of the fact that individuals using cordless phones, which generate comparable RF exposure to that from cell phones, was included in the “unexposed” category, and that among the four countries studied ORs for Denmark, Sweden and Switzerland were 1.73, 1.49 and 1.69, respectively, while that for Norway was 0.51. They suggest that this may reflect some methodological difference or bias.

f. Cardis E, Armstrong BK, Bowman JD, Giles GG, Hours M, et al. 2011. Risk of brain tumours in relation to estimated RF dose from mobile phones: results from five Interphone countries. *Occup Environ Med* 68:

631-640. ORs for tumours in the most exposed part of the brain in those with 10+ years of mobile phone use were 2.80 (1.13-6.94), and were significantly elevated after 7 years of use. The pattern for meningioma was similar but the ORs were lower.

g. Frei P, Poulsen AH, Johansen C, Olsen JH, et al. 2011. Use of mobile phones and risk of brain tumours: update of Danish cohort study. BMJ doi: 10.1136/bmj.d6387. Used the Danish cancer registry of 3.8 million persons. There were 10,729 cases of brain cancer between 1990-2007. No increased risk of brain tumors were found among cell phone subscribers as compared to non-subscribers. However, cordless phone subscribers were treated as non-cell phone users in this study.

h. Carlberg M, Hardell L. 2012. On the association between glioma, wireless phones, heredity and ionizing radiation. Pathophysiology 19: 243-252. Reports on two case-control studies of 1148 glioma cases. They find an OR = 2.9 (1.8-4.7) for ipsilateral use of mobile phones for more than ten years. For use of cordless phones they find an OR = 3.8 (1.8-8.1) for ipsilateral use for more than 10 years. ORs were higher for high grade gliomas. Risks were highest among those under age 20.

There are several reports investigating rates of cancer, particularly leukemia, in persons living near to AM or FM radio transmission towers or cell towers. While most of these studies report elevations in rates of cancer, their assessment of exposure is limited only to residential proximity to the towers, which is not a very exact monitor. None-the-less, these studies are significant because they directly monitor rates of human cancer. They also suggest that leukemia is the cancer of greatest concern when the whole body is exposed to radiofrequency radiation, in contrast to more localized cancers with localized exposure.

a. Michelozzi P, Capon A, Kirchmayer U, Forastiere F, Biggeri A, Barca A, Perucci CA. 2002. Adult and childhood leukemia near a high-power radio station in Rome, Italy. Am J Epidemiol 155: 1098-1103. The authors show that there is a significant elevation of childhood leukemia among residents living near to Vatican Radio (Standardized mortality ratio

1 = 2.2, 1.0-4.1), and that the risk declines with distance away from the
 2 transmitter (p = 0.03).

3
 4 b. Eger H, Hagen KU, Lucas B, Vogel P and Voit H. 2004. Einfluss
 5 der räumlichen Nähe von Mobilfunksendeanlagen auf die Krebsinzidenz.
 6 Umwelt-Medizin-Gesellschaft 17: 326-332. A German government-
 7 supported study of cancer risk in relation to residence close to cell towers
 8 found that rates were significantly higher (OR = 3.38, 95% CI = 1.39-8.25;
 9 99% CI = 1.05-10.91) for persons living within 400 m than among those
 10 living further away from the towers.

11
 12 c. Park SK, Ha M, Im HJ. 2004. Ecological study on residences in the
 13 vicinity of AM radio broadcasting towers and cancer death: preliminary
 14 observations in Korea. *Int Arch Occup Environ Health*. 77:387-394. This
 15 study found higher mortality areas for all cancers and leukemia in some age
 16 groups in the area near the AM towers.

17 d. Ha M, Im H, Lee M, Kim HJ, Kim BC, Gimm YM, Pack JK. 2007.
 18 Radiofrequency radiation exposure from AM radio transmitters and
 19 childhood leukemia and brain cancer. *Am J Epidemiol* 166: 270-279.
 20 Leukemia and brain cancer in children in Korea were investigated in
 21 relation to residence within 2 km of AM radio transmitters. There was a
 22 significant elevation in rates of leukemia (OR = 2.15, 1.00-4.67), but not of
 23 brain cancer in relation to peak, but not total radiofrequency exposure for
 24 children living within 2 km as compared to more than 20 km from the
 25 transmitters.

26
 27 e. Merzenich H, Schmiedel S, Bannack S, Bruggemeyer H, Phillipp J,
 28 et al. 2008. Childhood leukemia in relation to radio frequency
 29 electromagnetic fields in the vicinity of TV and radio broadcast
 30 transmitters. *Am J Epidemiol* 168: 1169-1178. Studied 1,959 cases of
 31 leukemia and 5,848 controls in Germany. They did not find any significant
 32 relationship between risk of leukemia and living within 2 km of a broadcast
 33 transmitter as compared to those living 10-15 km away.

34
 35 f. Elliott P, Toledano MB, Bennett J, Beale L, Best N, Briggs DF.
 36 2010. Mobile phone base stations and early childhood cancer: case-control
 37 study. *BMJ* 340: c3077 doi:10.1136/bmj/c3077. No association was found
 38 between risk of early childhood cancers and estimates of mother's exposure
 39 to mobile phone base stations during pregnancy.

40
 41 g. Dode AC, Leao M, Tejo FdeAF, Gomes ACR, Dode DC, Dode MC,
 42 Moreira CW, Condessa VA, Albinatti C and Calaffa WT. 2011. Mortality

1 by neoplasia and cellular telephone base stations in the Belo Horizonte
2 municipality, Minas Gerais State, Brazil. Sci Total Environ 409: 3649-
3 3665. This study shows higher rates of death from cancer among
4 individuals living close to cell towers than among those living further away.
5 Rates were highest in residences less than 100 m, falling to near
6 background a 1,000 m.

7
8 In summary, the ten major meta-analyses/pooled analyses, the recent cell phone
9 exposure studies, and the radio transmission exposure studies provide convincing
10 evidence of adverse health effects in humans associated with low-level RF
11 exposure. Other relevant evidence of human health effects is discussed in
12 Sections 11 and 12 of the *Bioinitiative Report* 2012.

13 Q. Is there evidence about the mechanisms by which low-level RF may adversely
14 affect human physiology?

15 Some, especially those from the physics and engineering community, are skeptical
16 of the ability of radiofrequency radiation to alter human physiological functions
17 because of the low energy of the non-ionizing portion of the electromagnetic
18 spectrum. The studies listed below provide evidence that cell phone use and
19 applied low-level radiofrequency radiation alter the metabolism of the brain and
20 various clinical measures in humans. They report a variety of effects on humans
21 including dose-dependent changes in cortisol and alpha-amylase, increased brain
22 glucose metabolism, chronic dysregulation of the catecholamine system, and
23 decreases in ACTH, cortisol, thyroid hormones, and prolactin in young females
24 and testosterone in males.

25 a. Augner C, Hacker GW, Oberfeld G, Florian M, Hitzl W, Hutter J,
26 Pauser G. 2010. Effects of exposure to base station signals on salivary

1 cortisol, alpha amylase and immunoglobulin A. Biomed Environ Sci
2 23:199-207. This was a human experimental study with exposure to pulsed
3 wave microwave radiation wherein immune indicators were monitored after
4 five 50-minute sessions. The researchers found dose-dependent changes in
5 cortisol and alpha-amylase.
6

7 b. Volkow ND, Tomasi D, Wange GJ, Vaska P, Fowler JS, Teland F,
8 Alexoff D, Logan J, Wong C. 2011. Effects of cell phone radiofrequency
9 signal exposure on brain glucose metabolism. JAMA 305:808-814. In
10 healthy participants and compared with no exposure, 50-minute cell phone
11 exposure was associated with increased brain glucose metabolism in the
12 region closest to the antenna. This shows direct effects of RF radiation on
13 the brain with cell phone use.
14

15 c. Buchner K, Eger H. 2011. Changes of clinically important
16 neurotransmitters under the influence of modulated RF fields – a long-term
17 study under real-life conditions. Umwelt-Medizin-Gesellschaft 24:44-57.
18 There was clear evidence of health-relevant effects, including an increase in
19 adrenaline and noradrenaline, and a subsequent decrease in dopamine in
20 people living near to a new MW-emitting base station. Levels of
21 phenylethylamine decreased and remained decreased, indicating chronic
22 dysregulation of the catecholamine system. Clinically documented
23 increases in sleep problems, headaches, dizziness, concentration problems
24 and allergies followed the onset of new microwave transmissions.
25

26 d. Eskander EF, Estefan SF, Abd-Rabou AA. 2011. How does long
27 term exposure to base stations and mobile phones affect human hormone
28 profiles? Clin Biochem 45:157-161. Measured hormone levels in 82
29 mobile phone users and 20 controls over a period of 6 years. Report that
30 there were decreases in ACTH, cortisol, thyroid hormones, and prolactin in
31 young females and testosterone in males. There was no change in serum
32 progesterone in females, but in older females prolactin increased with
33 exposure. Exposure from cell phone base stations was associated with
34 significant decreases in ACTH and cortisol.
35

36 The following studies report changes in male fertility and reproductive systems
37 associated with cell phone and low-level RF exposure.
38

39 a. Wdowiak A, Wdowiak L, Wiktor H. 2007. Evaluation of the effect
40 of using mobile phones on male fertility. Ann Agric Environ Med 14: 169-
41 172. Among Polish males with an infertility problem there was “an
42 increase in the percentage of sperm cells of abnormal morphology
43 associated with duration of exposure to waves emitted by the GSM phone.

1 It was also confirmed that a decrease in the percentage of sperm cells in
2 vital progressing motility in the semen is correlated with the frequency of
3 using mobile phones.”
4

5 b. Agarwal A, Deepinder F, Sharma RK, Ranga G, Li J. 2008. Effect
6 of cell phone usage on semen analysis in men attending infertility clinic: an
7 observational study. *Fert Steril* 89: 124-128.. “Use of cell phones
8 decreases the semen quality in men by decreasing the sperm count,
9 motility, viability, and normal morphology. The decrease in sperm
10 parameters was dependent on the duration of daily exposure to cell phones
11 and independent of the initial semen quality.”
12

13 c. Baste V, Riise T, Moen BE. 2008. Radiofrequency electromagnetic
14 fields: male infertility and sex ratio of offspring. *Int J Epidemiol* 23:369-
15 377. This is a study of Norwegian Navy personnel chronically exposed to
16 RF fields on the job. The rates of infertility were related to level of
17 exposure in a dose-dependent fashion.
18

19 d. Agarwal A, Desai NR, Makker K, Varghese A, et al. 2009. Effects
20 of radiofrequency electromagnetic waves (RF-EMW) from cellular phones
21 on human ejaculated semen: an *in vitro* pilot study. *Fert Stert* 92: 1318-
22 1325. “Radiofrequency electromagnetic waves emitted from cell phones
23 may lead to oxidative stress in human semen. We speculate that keeping
24 the cell phone in a trouser pocket in talk mode may negatively affect
25 spermatozoa and impair male fertility.
26

27 e. LaVignera S, Condorelli RA, Vicari E, D’Adata R, Calogero AE.
28 2012. Effects of the exposure to mobile phones on male reproduction: A
29 review of the literature. *J Androl* 33: 350-356. Studies in animals and
30 humans show that “RF-EMR decreases sperm count and motility and
31 increases oxidative stress....The results showed that human spermatozoa
32 exposed to RF-EMR have decreased motility, morphometric abnormalities
33 and increased oxidative stress, whereas men using mobile phones have
34 decreased sperm concentration, decreased motility (particularly rapid
35 progressive motility), normal morphology and decreased viability. These
36 abnormalities seem to be directly related to the duration of the mobile
37 phone use.”
38

39 f. Avendaño C, Mata A, Sanchez Sarmiento CA, Doncel GF. 2012.
40 Use of laptop computers connected to internet through Wi-Fi decreases
41 human sperm motility and increases sperm DNA fragmentation. *Fert Steril*
42 97:39-45. In this study human sperm were exposed to Wi-Fi from a laptop,
43 and were found to show reduced motility after a 4-hour exposure. The

1 results are consistent with other publications (see Agarwal et al., 2008. Fert
2 Steril 89:124-128) that reported that those who use cell phone regularly
3 have reduced sperm count.
4

5 Other evidence of fertility and reproductive effects of low-level RF exposure is
6 discussed in Section 18 of the *Bioinitiative Report 2012*.

7 Q. Is there evidence that some people may become hyper-sensitive to low-level RF
8 and experience related adverse health effects?

9 Electrical hypersensitivity (EHS) is a syndrome of relatively non-specific
10 complaints that are reported to be associated with exposure to electromagnetic
11 fields. The major symptoms are headache, fatigue, tinnitus, disruption of sleep,
12 mental dullness and a general feeling of ill health. Whether or not EHS exists has
13 been widely debated. In spite of widespread reports that up to 10% of the
14 population may suffer from EHS, most studies in laboratories with blinded
15 exposures (ie., the subjects do not know whether or not the fields are applied) have
16 not demonstrated that persons reporting to be electrosensitive can correctly
17 distinguish when the fields are on. However, there is increasing evidence that
18 EHS does exist and can be a disabling condition for some particularly sensitive
19 persons, although evidence to date is certainly incomplete.

20 There has been only one report of a completely blinded study of an
21 electrosensitive individual that has documented the ability of this individual to
22 report symptoms (primarily headache) in the presence of an electromagnetic field:

23
24 a. McCarty DE, Carrubba S, Chesson AL, Frilor C, Gonzalex-Toledo
25 E, Marino AA. 2011. Electromagnetic hypersensitivity: Evidence for a

1 novel neurological syndrome. Internat J Neurosci 121: 670-676. In a
2 female physician who is electrosensitive, blinded application of
3 electromagnetic fields triggered temporal pain, headache, muscle twitching
4 and skipped heartbeats within 100 seconds of field application.

5
6 There are a number of other reports investigating the prevalence of symptoms in
7 areas near to sources and/or other measures of human response to electromagnetic
8 fields. There are many publications on this subject, and the following are
9 representative of both positive and negative studies:

10
11 a. Hietanen M, Hamalainen A-M, Husman T. 2002. Hypersensitivity
12 symptoms associated with exposure to cellular telephones: No causal link.
13 Bioelectromagnetics 23: 264-270. Studied 20 volunteers who reported
14 themselves to be electrosensitive and exposed them to fields in a blinded
15 manner. "None of the test subjects could distinguish real RF exposure from
16 sham exposures."

17
18 b. Abelin T, Altpeter E, Rösli M. 2005. Sleep disturbances in the
19 vicinity of the short-wave broadcast transmitter Schwarzenburg.
20 Somnologie 9:203-209. There is strong evidence of a causal relationship
21 between operation of a short-wave radio transmitter and sleep disturbances
22 in the surrounding population.

23
24 c. Hutter HP, Moshammer H, Wallner P, Kundi M. 2006. Subjective
25 symptoms, sleeping problems, and cognitive performance in subjects living
26 near mobile phone base stations. Occup Environ Med 63:307-313. There
27 was a significant relation of some symptoms, especially headaches, to
28 measured power density, as well as effects on wellbeing and performance.

29
30 d. Eliyahu I, Luria R, Hareuveny R, Margalit M, Neiran N, Shani G.
31 2006. Effects of radiofrequency radiation emitted by cellular telephones on
32 the cognitive functions of humans. Bioelectromagnetics 27:119-266. A
33 total of 36 human subjects were exposed to pulse-modulated microwaves
34 and were tested on four distinct cognitive tasks. Exposure to the left side of
35 the brain slows left-hand response time in three of the four tasks.

36
37 e. Altpeter ES, Rösli M, Battaglia M, Pfluger D, Minder CE, Abelin
38 T. 2006. Effect of short-wave magnetic fields on sleep quality and
39 melatonin cycle in humans: The Schwarzenburg shut-down study.

1 Bioelectromagnetics 27:142-150. Sleep quality improved and melatonin
2 excretion increased when the transmitter was shut down.

3
4 f. Preece AW, Georgious AG, Duunn EJ, Farrow SC. 2007. Health
5 response of two communities to military antennae in Cyprus. *Occup*
6 *Environ Med* 64:402-408. Compared to residents of a control village, there
7 was a highly significant excess in the reporting of migraine, headache and
8 dizziness in residents living near to military and cell phone antenna
9 systems.

10
11 g. Barth A, Winker R, Ponocny-Seliger E, Mayrhofer W, Ponocny I,
12 Sauter C, Vana N. 2008. A meta-analysis for neurobehavioural effects due
13 to electromagnetic field exposure emitted by GSM mobile phones. *Occup*
14 *Environ Med* 65: 342-345. The authors looked at 19 studies of cognitive
15 function in cell phone users, and found in the meta-analysis that there is
16 evidence for a decreased reaction time, altered working memory and
17 increased number of errors in exposed persons.

18
19 h. Landgrebe M, Frick U, Hauser S, Langguth B, et al. 2008.
20 Cognitive and neurobiological alterations in electromagnetic hypersensitive
21 patients: results of a case-control study. *Psychol Med* 38: 1781-1791.
22 Studies 89 EHS subjects and 107 age and gender matched controls. Found
23 that discrimination ability was significantly reduced in EHS subjects, while
24 intra-cortical facilitation was decreased in younger, but increased in older
25 EHS subjects. They conclude that there are significant cognitive and
26 neurobiological alterations pointing to a higher genuine individual
27 vulnerability in EHS subjects.

28
29 i. Landgrebe M, Frick U, Hauser S, Hajak G, Langguth B. 2009.
30 Association of tinnitus and electromagnetic hypersensitivity: hints for a
31 shared pathophysiology? *PLoS One* 4: e5026 doi:
32 10.1371/journal.pone.0005026. Tinnitus occurrence and severity were
33 assessed by questionnaire in 89 EHS and 107 control subjects. Tinnitus
34 was significantly more frequent in the EHS group, but there were no
35 differences in severity or duration. They conclude that tinnitus is
36 associated with subjective EHS.

37
38 j. Furubayashi T, Ushiyama A, Teerao Y, Mizuno Y, et al. 2009.
39 Effects of short-term W-CDMA mobile phone base station exposure on
40 women with or without mobile phone related symptoms.
41 *Bioelectromagnetics* 30: 100-113. In a double-blind, cross over study of 11
42 subjects with cell phone-related symptoms and 43 controls, subjected to
43 continuous, intermittent and sham exposure with or without noise, no

1 significant effects were found on any psychological, cognitive or autonomic
2 response.

3
4 k. Dahmen N, Ghezel-Ahmadi D, Engel A. 2009. Blood laboratory
5 findings in patients suffering from self-perceived electromagnetic
6 hypersensitivity (EHS). *Bioelectromagnetics* 30: 299-306. Monitored
7 thyroid hormone, liver enzymes, hemoglobin, hematocrit and c-reactive
8 protein in subjects with and without EHS. "Our results identified
9 laboratory signs of thyroid dysfunction, liver dysfunction and chronic
10 inflammatory processes in small, but remarkable fractions of EHS
11 sufferers."

12
13 l. Eger H, Jahn M. 2010. [Specific health symptoms and cell phone
14 radiation in Selbitz (Bavaria, Germany)- Evidence of a dose-response
15 relationship.] *Umwelt-Medizin-Gesellschaft* 23: 2. Reports on symptoms
16 of individuals based on residential location and RF measurements of local
17 cell phone radiation levels. "For symptoms as sleep problems, depressions,
18 cerebral symptoms, joint problems, infections, skin problems,
19 cardiovascular problems as well as disorder of the visual and auditory
20 systems and the gastrointestinal tract, a significant dose-response
21 relationship was observed in relation to objectively determined exposure
22 levels".

23
24 m. Robertson JA, Théberge J, Weller J, Drost DJ, Prato FS, Thomas
25 AW. 2010. Low-frequency pulsed electromagnetic field exposure can alter
26 neuro-processing in humans. *JR Soc Interface* 7:467-473. A functional
27 magnetic resonance imaging study demonstrated how the neuromodulation
28 effect of extremely low-frequency magnetic fields influences the processing
29 of acute thermal pain. The study concludes that magnetoreception may be
30 more common than presently thought. This study was already filed in the
31 present case as Exhibit C-SE-AQLPA-0043, SE-AQLPA-5, Document 10.

32
33 n. Heinrich S, Thomas S, Heumann C, von Kries R and Radon K.
34 2010. Association between exposure to radiofrequency electromagnetic
35 fields assessed by dosimetry and acute symptoms in children and
36 adolescents: a population based cross-sectional study. *Environ Health* 9: 75
37 doi: 10.1186/1476-069X-9-75. The authors studied 1484 children and 1508
38 adolescents with radiofrequency exposure monitored by a personal
39 dosimeter. Self-reported statistically significant effects found include
40 increased headache (OR 1.50, 1.03-2.19), greater irritation in the evening
41 (OR 1.79, 1.23-2.61) and higher concentrations problems (OR = 1.55, 1.02-
42 2.33) in individuals with greater exposures. However, many others
43 measures did not lead to statistically significant associations.

1
2 o. Mohler E, Frei P, Braun-Fahrlander C, Frohlich J, et al. 2010.
3 Effects of everyday radiofrequency electromagnetic field exposure on sleep
4 quality: A cross-sectional study. *Rad Res* 174: 347-356. Studied 1375
5 inhabitants of Basel with a questionnaire and using a prediction model of
6 exposure. "Neither mobile phone use nor cordless phone use was
7 associated with decreased sleep quality."

8
9 p. Roosli M, Frei P, Mohler E, Hug K. 2010. Systematic review on the
10 health effects of exposure to radiofrequency electromagnetic fields from
11 mobile phone base stations. *Bull World Health Organ* 88: 887-896.
12 Reviewed 17 publications on non-specific symptoms of ill health from RF
13 exposure from mobile phone base stations, and concluded that "At present
14 there is insufficient data to draw firm conclusions about health effects from
15 long-term low-level exposure typically occurring in the everyday
16 environment."

17
18 q. Papageorgiou CC, Hountala CD, Maganioti AE, Kyprianou MA,
19 Rabavilas AD, Papadimitriou GN, Capsalis CN. 2011. Effects of wi-fi
20 signals on the p300 component of event-related potentials during an
21 auditory Hayling task. *J Integr Neurosci* 10:189-202. The Hayling
22 Sentence Completion test was used to evaluate response initiation and
23 response inhibition. This study concludes that WI-FI exposure may exert
24 gender-related alterations on neural activity.

25
26 r. Oshima N, Nishida A, Shimodera S, Tochigi M, et al. 2012. The
27 suicidal feelings, self-injury, and mobile phone use after lights out in
28 adolescents. *J Pediatr Psychol* 37: 1023-1030. Studied 17,920 adolescents
29 using a self-report questionnaire. "Logistic regression showed significant
30 associations of the nocturnal mobile phone use with poor mental health,
31 suicidal feelings, and self-injury after controlling for sleep length and other
32 confounders."... "A mechanism of the association might be worsening of
33 the quality of sleep."

34
35 In summary, some studies are suggestive of an association, but the reported
36 evidence falls short of proof. In the context of exposure to RF emissions from
37 smart meters, there is a substantial body of evidence from the personal accounts of
38 utility customers who report experiencing EHS symptoms. This evidence should

1 not be disregarded in setting public policy that will determine whether and to what
2 extent people are exposed to these devices.

3 Further discussion of studies of EHS effects can be found in Sections 6 and
4 8 of the Bioinitiative Report 2012.

5 Q. Is there evidence that brain cancer rates have increased in recent decades?

6 A. If use of cell phones causes brain cancer, then one might expect that overall rates
7 of brain cancer would show an increase, since cell phone use has grown
8 enormously in recent years. However, since use of cell phones is relatively recent
9 and the latency for development of brain cancer following other environmental
10 exposures is long (up to 20-30 years), there might not yet be a clear pattern of
11 increased incidence. The following studies address this issue:

12
13 a. Central Brain Tumor Registry of the United States (CBTRUS).
14 Supplemental Report: Primary Brain Tumors in the United States, 2004.
15 Hinsdale, IL; Central Brain Tumor Registry of the United States 2008.
16 Age-adjusted CNS tumor incidence was 18.2 cases per 100,000 in 2004,
17 but 13.4 cases per 100,000 in 1995.

18
19 b. Lehrer S, Green S, Stock RG. 2010. Association between number of
20 cell phone contracts and brain tumor incidence in nineteen U.S. states. J
21 Neuro-Oncol 101:505-507. "The effect of cell phone subscriptions was
22 significant ($P = 0.017$), and independent of effect of mean family income (P
23 $= 0.894$), population ($P = 0.003$) and age (0.499). The very linear
24 relationship between cell phone usage and brain tumor incidence is
25 disturbing and certainly needs further epidemiological evaluation. In the
26 meantime, it would be prudent to limit exposure to all source of electro-
27 magnetic radiation."

28
29 c. De Vocht F, Burstyn I, Cherrie JW. 2011. Time trends (1998-2007)
30 in brain cancer incidence rates in relation to mobile phone use in England.
31 Bioelectromagnetics 32:334-339. "There were no time trends in overall
32 incidence of brain cancers for either gender, or any specific age groups.

1 Systematic increases in rates for cancer of the temporal lobe in men... and
2 women... were observed, along with decreases in the rates of cancer of the
3 parietal lobe... and cerebellum...”

4
5 d. Little MP, Curtis RE, Devesa SS, Inskip PD, et al. 2012. Mobile
6 phone use and glioma risk: comparison of epidemiological study results
7 with incidence trends in the United States. *BMJ* 344: e1147 doi:
8 10.1136/bmj.e1147. “Raised risks of glioma with mobile phone use, as
9 reported by one (Swedish) study forming the basis of the IARC’s re-
10 evaluation of mobile phone exposure, are not consistent with observed
11 incidence trends in US population data, although US data could be
12 consistent with the modest excess risks in the Interphone study.”

13
14 e. Dobes M, Shadbolt B, Khurana VG, Jain S, et al. 2011. A
15 multicenter study of primary brain tumor incidence in Australia (2009-
16 2008). *Neuro-Oncol* 13: 783-790. The authors observed an increased
17 increase in malignant primary brain tumors over the period 2000-2008, but
18 cannot determine whether it was due to improved detection, diagnosis or to
19 a true elevated incidence.

20
21 f. Deltour I, Auvienne A, Feychting M, Johansen C, et al. 2012. Mobile
22 phone use and incidence of glioma in the Nordic countries 1979-2008.
23 *Epidemiology* 23:301-307. “No clear trend change in glioma incidence
24 rates was observed. Several of the risk increases seen in case-control
25 studies appear to be incompatible with the observed lack of incidence rate
26 increase in middle-aged men. This suggests longer induction periods than
27 currently investigated, lower risks than reported from some case-control
28 studies, or the absence of any association.”

29
30 g. The Danish Cancer Society recently reported that the number of men
31 who are diagnosed with the most malignant form of brain cancer
32 (glioblastoma) has almost doubled over the past ten years.
33 ([http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i](http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i+hjernesvulster.htm)
34 [+hjernesvulster.htm](http://www.cancer.dk/Nyheder/nyhedsartikler/2012kv4/Kraftig+stigning+i+hjernesvulster.htm))

35
36 Further discussion of the relevance of brain cancer rates to the debate about the
37 association between cell phone and RF exposure to cancer is found in Section 11
38 of the *Bioinitiative Report, 2012*.

1 Q. In addition to the foregoing evidence of the effects of low-level RF on humans, is
2 there additional evidence from studies of animals and isolated cells?

3 A. Some, but not all studies of isolated cells and intact animals have shown that
4 RF/MW exposures may cause changes in cell membrane function, cell
5 communication, metabolism, activation of proto-oncogenes, and can trigger the
6 production of stress proteins at exposure levels below the above FCC and Health
7 Canada guidelines. Resulting effects in cellular studies include DNA breaks and
8 chromosome aberrations, cell death including death of brain neurons, increased
9 free radical production, activation of the endogenous opioid system, cell stress and
10 premature aging. Additional studies show neurologic, immune, endocrine,
11 reproductive and cardiac, adverse health effects from low-dose, chronic exposure
12 to RF/MW radiation in humans. These studies will not be presented here because
13 there are too many and their relevance to human health is uncertain. Please see
14 Bioinitiative Report, 2012 for a comprehensive review of these studies. In
15 summary they do provide additional evidence of biological effects and evidence
16 for possible mechanisms whereby radiofrequency fields may cause adverse health
17 effects including cancer, reproductive and neurobehavioral effects through
18 generation of reactive oxygen species, gene induction and alteration of ion fluxes,
19 but not all positive observations have been fully replicated.

20 **Q. Are there any safety standards or guidelines governing RF devices in the**
21 **United States that are designed to protect people from non-thermal effects of**
22 **RF exposure?**

1 A. The standards set by the US Federal Communications Commission (FCC)
2 and most international government and non-government organizations are based
3 on the fallacious assumption that there are no adverse human health effects from
4 radiofrequency radiation that does not cause measureable heating. These
5 standards provide no protection whatsoever against non-thermal effects of RF.
6 Some biological effects are known to occur at several hundred thousand times
7 below the FCC public exposure guidelines and the similar guidelines of Health
8 Canada's Safety Code no. 6 (of 6,000,000 $\mu\text{W}/\text{m}^2$ or 600 $\mu\text{W}/\text{cm}^2$ for the 902-928
9 MHz bandwidth), as documented in the 2012 *Biointiative Report*, Section 24. It
10 is further to be noted that FCC guidelines also apply to 30-minute averaging and
11 Health Canada's Safety Code no. 6 applies to 6-minute averaging. There is no
12 evidence that averaging exposures over time is appropriate for assessing maximum
13 exposure limits to low-level RF.

14 Furthermore, these limits are based on the incorrect biological assumption
15 that body temperatures must increase at least 1°C to lead to potential biological
16 impacts and the impacts of absorbing RF within the band of the electromagnetic
17 spectrum that smart meters use would only be limited to behavioral disruption.
18 These limits do not take into account the scientific research that show tissue
19 heating may result in many adverse health effects other than "behavioral
20 disruption". These limits also do not take into account the accepted biological fact
21 that every enzyme system in the body is exquisitely sensitive to temperature and
22 may increase activity by even a fraction of a degree increase in temperature. What

1 is defined as “non-thermal” effect is therefore partly a function of our ability to
2 measure the temperature increase. *See Bioinitiative Report*, Section 24 for further
3 discussion.

4 FCC public RF/MW radiation exposure guidelines (and the similar Health
5 Canada Safety Code no. 6 guidelines) are based on the height, weight and stature
6 of a 6-foot tall man, not children or adults of smaller stature. The guidelines do not
7 take into account the unique susceptibility of growing children to RF/MW
8 radiation exposures. Since children are growing, their rate of cellular activity and
9 division is more rapid, and they are at a greater risk for DNA damage and
10 subsequent cancers. Growth and development of the central nervous system is still
11 occurring well into the teenage years, such that the neurological impairments
12 predictable by the extant science may have great impact upon development,
13 cognition, learning, and behavior.

14 **Q. Have you reviewed the joint testimony of William H. Bailey, Ph.D. and Yakov**
15 **Shkolnikov, Ph.D., dated September 19, 2012?**

16 A. Yes.

17 **Q. In their testimony, Dr. Bailey and Dr. Shkolnikov cite a report by the**
18 **ICNIRP Committee, which concluded that “the trend in the accumulated**
19 **evidence is increasingly against the hypothesis that mobile phone use causes**
20 **brain tumors.” Do you agree with that conclusion?**

21 A. I strongly disagree. The weight of evidence indicates that mobile phone use is
22 associated with elevated risk of brain cancer which becomes apparent after ten or

1 more years of intensive use and occurs primarily on the side of the head where the
2 user holds his/her phone the majority of the time. There is emerging evidence that
3 younger people are at greater risk than older individuals. The great majority of the
4 meta-analyses that have been published on the subject demonstrate a statistically
5 significant elevation in rates of brain cancer with long-term cell phone use. This
6 statement by Bailey and Shkolnikov is simply not true.

7 It is necessary to comment on the ICNIRP report, as well as on the UK
8 Advisory Group on Non-Ionising Radiation (AGNIR) report, published in April,
9 2012, which is also cited by Bailey and Shkolnikov. It should be noted that there
10 is considerable overlap in the membership of these two groups. Both ignore or
11 attempt to discredit the information presented above. The AGNIR report fails to
12 even mention the IARC classification of radiofrequency fields as possible human
13 carcinogens. Neither is a fair and balanced review of the scientific evidence
14 concerning the human health effects of radiofrequency fields. A much more
15 convincing review of the evidence is found in the Ramazzini Institute European
16 Journal of Oncology Library, Volume 5, entitled "Non-thermal effects and
17 mechanisms of interaction between electromagnetic fields and living matter,"
18 published in 2010, and in the *Bioinitiative Report, 2012*. The primary reason that I
19 and the other authors prepared the *Bioinitiative Report* was and is to counter the
20 prejudicial and false conclusions of these reports, and to do so by presenting a
21 comprehensive review of scientific evidence.

1 Q. Do you agree with their testimony that the authors of the *Bioinitiative Report*
2 used flawed methods and failed to follow “the standard, scientific methods for
3 developing exposure limits.”

4 A. I strongly disagree with this statement. It should be noted that the *Bioinitiative*
5 *Report* does not recommend exposure limits *per se*, but rather identifies exposures
6 levels which are associated with biological effects, some of which are adverse
7 effects on human health. The public health chapter, of which I am a co-author,
8 identifies a “no observed effect level” (NOEL), based on the scientific evidence
9 from peer-reviewed scientific studies, then applies safety factors for sensitive
10 populations (the fetus, children, the aged, etc.) as is standard practice in chemical
11 risk assessment. This chapter presents clear documentation of why more stringent
12 limits on exposure are necessary to protect human health.

13 The *BioInitiative Report* is aimed at *restoring* the balance, by providing a
14 more comprehensive review of the evidence. The *Bioinitiative Report* mentions
15 many negative reports, discusses the weight of evidence, and looks for
16 inconsistencies. For example, Prof. Henry Lai of the University of Washington in
17 the 2012 *Bioinitiative Report* presents summaries of 86 scientific studies on
18 genotoxic effects of radiofrequency radiation published since 2007, and finds that
19 63% of these found statistically significant positive effects, while of 155 new
20 studies on neurological effects, 98 found effects. The *Bioinitiative Report*, unlike
21 either the ICNIRP or AGNIR reports, reviews of the scientific research available,
22 both those showing and not showing biological effects and human disease, and

1 draws conclusions based on the weight of the evidence that standard setting
2 organizations were failing to properly take into account.

3
4 **Q. Dr. Bailey and Dr. Shkolnikov testified that: “The weight of the evidence does**
5 **not support the idea that significant biological or adverse health effects can**
6 **occur” from RF exposure. Do you agree with this conclusion?**

7 A. This statement is almost incomprehensible given the strength of the evidence
8 demonstrating consistent and serious adverse health effects in both animal and
9 human studies. The studies of greatest importance are those which demonstrated
10 elevations in cancers, especially leukemia and brain cancer, in association with
11 exposure to radiofrequency EMFs. There is evidence that exposure to cell phone
12 frequencies increased uptake of glucose in the brain, which indicates that RF
13 radiation alters fundamental process within the nervous system. The thousands of
14 studies in cellular and animal systems provide additional evidence that
15 radiofrequency fields alter a host of biochemical, physiological and behavioral
16 factors. While certainly not every study reports positive and statistically
17 significant results, the majority do as clearly documented in the 2012 *Bioinitiative*
18 *Report*. No objective person could possibly make a statement such as this if they
19 are at all familiar with the literature published in high-quality, peer-reviewed
20 scientific journals, and if they are coming to the question with an open mind
21 without a major conflict of interest.

1 Standards setting organizations aimed at regulating RF exposure have for a
2 long time been dominated by physicists and engineers, often with close ties with
3 the industry, with little input from biological and medical science. In spite of
4 evidence to the contrary, many such people have as a statement of faith that RF
5 fields that do not cause measureable tissue heating cannot have biologic effects.
6 This point of view is incompatible with the science. Standards setting
7 organizations also often explicitly take into account the economic impacts of the
8 standards when faced with scientific uncertainty. Both because of their training
9 and because of their ties with the industry, members of most of these organizations
10 have been reluctant to take the above biological findings into account when
11 proposing exposure limits.

12 These organizations have generally refused to accept epidemiological and
13 laboratory research findings linking RF electromagnetic fields exposure with
14 various non-thermal biological effects, as being inconclusive and requiring further
15 research. The difficulty stems from the fact that, although links have been
16 demonstrated repeatedly between RF electromagnetic fields exposure and non-
17 thermal biological effects in humans, there is a lack of a comprehensive biological
18 theory explaining why these effects take place, and therefore causality cannot, at
19 the present time, be demonstrated with certainty. Animals do not always respond
20 to RF electromagnetic fields as do humans. Also, in some cases, experimental
21 results in cellular studies have not been replicated in other laboratories; in some
22 cases attempts to duplicate results showed negative results or variations in the

1 results. These discrepancies are, however, normal in the research process and may
2 result from slight, but significant differences in procedures; they indicate that
3 biological systems are complex and that different variables need to be isolated in
4 order to fully understand these systems. Research is still needed in order to
5 determine to what extent non-thermal biological effects may vary with frequency,
6 with modulation and depend on the pulsed (instead of continuous) character of RF
7 emissions. There may also be variance between the levels of reaction of different
8 subjects for reasons that still remain to be explained. This is what the research
9 process is about. In biology and medicine there is nothing that is 100% proven;
10 our understanding of various illnesses, cancer and Alzheimer's, for example, is
11 still largely incomplete. We rely on statistical significance and weight of evidence
12 and, therefore, on judgment, when drawing conclusions about health effects.

13 **Q. In your opinion, could a careful scientist familiar with the body of knowledge**
14 **on the subject reliably conclude that there are no risks of adverse health**
15 **effects from the exposure to RF in the 2.4 GHz range?**

16 A. On the basis of the vast body of scientific literature, many public health experts,
17 myself included, are of the opinion that exposure to RF/MW radiation and EMFs,
18 including in the range of 2.4GHz, poses a potential of serious threat to public
19 health. The degree of risk will vary with both the intensity and duration of
20 exposure. It is likely society will face markedly increased incidence of neurotoxic
21 effects, neurodegenerative diseases, cancers and genotoxicity in the future,

1 resulting from the extreme and mostly involuntary exposure to RF/MW radiation
2 and EMFs.

3 **Q. Are you familiar with smart meter technology?**

4 A. I am familiar with smart meter technology.

5 **Q. In your opinion, could a careful scientist familiar with the body of knowledge**
6 **on the subject reliably conclude that there are no risks of adverse health**
7 **effects from exposure to RF from smart meters emitting RF radiation in the**
8 **2.4 GHz range with peak power densities of approximately 0.44 mW/cm²?**

9 A. There are two types of smart meter technology. Wired smart meters pose no risk
10 of exposure to RF radiation. Wireless smart meters, on the other hand, pose a
11 substantial risk of RF exposure which is dependent on the frequency of pulsed RF,
12 the intensity of the pulsed RD and the individual's distance from the meter. While
13 there have not been human health studies done to date of the effects of exposure to
14 smart meter RF, because the technology is too new and the latency for adverse
15 effects for diseases such as cancer is long, the evidence from the cell phone studies
16 demonstrates convincingly that wireless smart meters pose a risk to human health.

17 Smart meters send pulsed RF radiation at intermittent periods of time.
18 While the frequency of these pulses may vary with different smart meters, some
19 have been reported to send pulses over 30 times a minute at peak power density
20 reading of over 67mW/m² (0.0067mW/cm²) (Maisch. 2012. Smart meter health
21 concerns: Just a nocebo effect or an emerging public health nightmare? ACNEM
22 Journal 31: 15-19), and this exposure has been associated with self-reported

1 experimental studies that provide some of the evidence of low intensity exposure
2 effects from radiofrequency radiation at low-intensity exposures. Because the
3 meters operate intermittently 24/7, an individual in the vicinity of the meter will be
4 continuously exposed to RF.

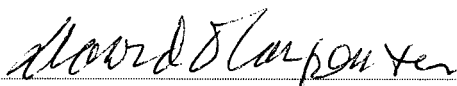
5 It is correct that the CMP smart meters comply with the FCC standard of 1
6 mW/cm². The problem is that the FCC standard is based on the assumption that
7 there are no effects of RF radiation other than tissue heating, which is simply not
8 the case.

9 For most smart meter use, the cumulative average RF exposure is not great,
10 but the reported health effects are large. This raises the important question as to
11 whether the exposure of greatest concern is the cumulative average, or rather the
12 peak power levels in the pulses. This issue is discussed in Chapter 24 of the 2012
13 *Biointiative Report*, which presents some evidence that it is the peak power that is
14 important. However, the total exposure will only increase in the future as RF
15 devices are being placed in every appliance in the home, and will use RF to
16 communicate to the smart meter which will communicate with the utility. This
17 will make the home, especially the kitchen, a source of highly elevated RF
18 exposure whenever an appliance is used.

19 Further investigation of the human health effects of smart meter exposures
20 is essential. In the meantime it is extremely unwise to implement the smart grid
21 with wireless smart meters until we understand fully the potential for harm to
22 human health.

1

Dated this 18th day of January, 2013.

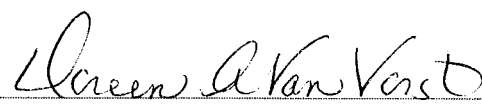


David O. Carpenter, M.D.

STATE OF NEW YORK
RENSSELAER, ss:

January 22, 2013

Personally appeared the above-named David O. Carpenter, M.D., and stated under oath that the foregoing Affidavit made by him is true and based upon his own personal knowledge, information or belief, and so far as upon information and belief, he believes the information to be true. Before me,



Notary Public/Attorney-at-Law
Doreen A Van Vorst

Name Typed or Printed
My Commission Expires: _____

DOREEN A. VanVORST
Notary Public, State of New York
Qualified in Rensselaer County
Reg. No. 01VA5083834
My Commission Expires Aug. 25, 2013

**DAVID CARPENTER
EXHIBIT A**

CURRICULUM VITAE

Name: David O. Carpenter

Home Address: 2749 Old State Road
Schenectady, New York 12303

Positions Held:
Director, Institute for Health and the Environment
University at Albany
Professor, Environmental Health Sciences
School of Public Health, University at Albany
5 University Place, A217, Rensselaer, NY 12144

Honorary Professor
Queensland Children's Medical Research Institute
University of Queensland
Brisbane, Australia

Education: 1959 B.A., Harvard College, Cambridge, MA
1964 M.D., Harvard Medical School, Boston, MA

Positions Held:

9/61-6/62 Research Fellow, Department of Physiology, University of Göteborg, Sweden with Professor Anders Lundberg

7/64-6/65 Research Associate, Department of Physiology, Harvard Medical School, Boston, MA under the direction of Dr. Elwood Henneman

7/65-2/73 Neurophysiologist, Laboratory of Neurophysiology, National Institutes of Mental Health, Dr. Edward V. Evarts, Chief, Assistant Surgeon, USPHS, currently a Reserve Officer in the USPHS.

2/73-3/80 Chairman, Neurobiology Department Armed Forces Radiobiology Research Institute, Defense Nuclear Agency, Bethesda, MD

3/80-9/85 Director, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY

9/85-1/98 Dean, School of Public Health, University at Albany

9/85-Pres. Professor, Departments of Environmental Health Sciences and Biomedical Sciences, School of Public Health, University at Albany.

9/85-7/98 Research Physician, Wadsworth Center for Laboratories and Research, New York State Department of Health, Albany, NY

1/98-1/05 Adjunct Professor in the Center for Neuropharmacology & Neuroscience, Albany Medical College, Albany, NY

2001-Pres. Director, Institute for Health and the Environment, University at Albany, SUNY, Rensselaer, NY. The Institute was named a Collaborating Center of the World Health Organization in 2011.

2005-Pres. Senior Fellow, Alden March Bioethics Institute, Albany Medical College/Center, Albany, New York

DAVID CARPENTER **EXHIBIT A**

Editor-in-Chief: Cellular and Molecular Neurobiology, 1981 – 1987
Editor-in Chief: Reviews on Environmental Health 2012-present
Editor-in-Chief: Journal of Local and Global Health Sciences 2012-present
Editorial Advisor: Cellular and Molecular Neurobiology, 1987 - Present
Editorial Boards: Journal of Public Health Management and Practice, 1995 - 2002
International Journal of Occupational Medicine & Environmental Health
1996 – Present
Journal of Alzheimer's Disease – Associate Editor, 2007-2009
Reviews in Environmental Health; 2008-2012
International Archives of Occupational and Environmental Health; 2009-present.
Journal of Environmental and Public Health, 2009-present.
Environmental Health Perspectives, 2010-present
Global Health Perspective, 2012-present

National and International Committees:

1978, 1981 Physiology Study Section (Ad hoc member)
1979-1985 NIH International Fellowship Study Section
1974-1981 Member, Steering Committee of the Section on the Nervous System, American Physiological Society (Chairman of the Committee, 9/76-4/80)
1981-1989 Member, USA National Committee for the International Brain Research Organization
1985-1986 Committee on Electric Energy Systems of the Energy Engineering Board, National Research Council
1986-1987 Member, Neurophysiology Peer Panel for the National Aeronautics and Space Administration
1987-1989 Member, Science Advisory Council of the American Paralysis Association
1987-1990 Advisory Panel for the Electric Energy System Division, U.S. Department of Energy
1985-1993 Committee #79, National Council on Radiation Protection and Measurements
1986-1997 Member, Legislative and Education Committees, Association of Schools of Public Health
1989-1994 Member, Neuroscience Discipline Working Group, Life Sciences Division of the NASA
1994, 1995 Federation of American Societies for Experimental Biology Consensus Conference on FY 1995 Federal Research Funding
1994-1997 Member, Legislative Committee of the Association of Schools of Public Health
1997 Member, Executive Committee of the Association of Schools of Public Health
1997-2000 National Advisory Environmental Health Sciences Council of the National Institutes of Health
1998-Pres. Member, U.S. Section of the Great Lakes Science Advisory Board of the International Joint Commission
2000-Pres. Member, Board of Directors, Pacific Basin Consortium for Hazardous Waste Health and Environment; Treasurer, 2001-2004, 2008-pres; Chair, 2004-2008
2001-2008 United States Co-Chair, Workgroup on Ecosystem Health of the Science Advisory Board of the International Joint Commission
2002-2003 Member, Committee on the Implications of Dioxin in the Food Supply, The National Academies, Institute of Medicine
2003-2008 Member, United States Environmental Protection Agency, Children's Health Protection Advisory Committee
2003-Pres. Chair, Advisory Committee to the World Health Organization and National Institute of Environmental Health Sciences on collaborative activities.
2004-Pres. Member, Blue Ocean Institute Curriculum Advisory Board.
2007-2011 Chair, Workgroup on Risks vs. Benefits of Fish Consumption, Science Advisory Board, International Joint Commission.

DAVID CARPENTER EXHIBIT A

State and Local Committees:

1980-1987	Executive Secretary, New York State Power Lines Project
1985-1989	Board of Scientific Advisors, Institute of Basic Research, OMRDD, N.Y.
1986-1989	Member, Steering Committee, Health Policy and Administrative Consortium of the Capital District
1991-1992	Member, Connecticut Academy of Sciences and Engineering Committee on Electromagnetic Field Health Effects
1991-1992	Member, Board of Directors of the Capital District Chapter of the Alzheimer's Disease and Related Disorders Association, Inc.
1991-1992	Member, State Task Force for the Reform of Middle Level Education in NY State
1992-1993	Member, State Needs Task Force on Health Care and Education
1987-1998	Delegate-at-Large, New York State Public Health Association
1991-1995	Member, Board of Directors of the Capital District Amyotrophic Lateral Sclerosis Association
1994	Chair, Council of Deans, University at Albany, SUNY
1997-2008.	Member, Board of Directors, (Chair 1998-2004) Albany-Tula Inc.: A Capital Region Alliance
2000-Pres.	Member, Board of Directors, Healthy Schools Network, Inc.
2000-2003	Member, Medical Advisory Board, Hepatitis C Coalition, New York
2000-2004	Member, Environmental Protection Agency /National Association of State Universities and Land Grant Colleges Task Force
2001-2008	Member, Board of Directors, Environmental Advocates of New York
2004-2007	Member, Ad Hoc Advisory Group on Brownfield Cleanup Standards
2005-Pres.	Member, Schooling Chefs Curriculum Advisory Board
2005-Pres.	Member, Advisory Board, Healthy Child Healthy World
2005-2008	Member, Board of Directors, Citizens Environmental Coalition
2006-2009	Member, Board of Directors, Marine Environmental Research Institute
2007-2009	Member, New York State Renewable Energy Task Force

Honors, Awards and Fellowships:

1959	B.A. awarded <u>magna cum laude</u> . Thesis entitled "Metamorphosis of visual pigments: A study of visual system of the salamander, <u>Ambystoma tigrinum</u> " (Thesis advisor, Professor George Wald) Elected to Phi Beta Kappa and to Sigma Xi
1964	M.D. awarded <u>cum laude</u> for a thesis in a special field. Thesis entitled "Electrophysiological observations on the importance on neuron size in determining responses to excitation and inhibition in motor and sensory systems" (Thesis advisor, Dr. Elwood Henneman)
1964	Awarded the Leon Resnick Prize given to a Harvard Medical School graduate showing promise in research
1970	Awarded the Moseley Traveling Fellowship for study in England (Fellowship declined)
1971	Invited as Visiting Professor of Physiology, Centro de Investigacion y de Estudios Avanzados, del Institute Politecnico Nacional, Mexico 14, D.F., Mexico, for 3 months
1982, 1986	Visiting Professor of Physiology, Department of Physiology, Kyushu
1987	University, Fukuoka, Japan, for a period of three months each
1989	Awarded Jacob Javits Neuroscience Investigator Award from the National Institute of Neurological and Communicative Diseases and Stroke
1999	Awarded Homer N. Calver Award from the American Public Health Association for studies

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- in environmental health.
- 2001 Awarded 2001 Academic Laureate from the University at Albany Foundation.
- 2010 Awarded the Albion O. Bernstein, M.D. Award in recognition of an outstanding contribution to public health and the prevention of disease through lifelong research of environmental health hazards and for limitless devotion to medical education by the Medical Society of the State of New York.
- 2011 Awarded the Rodney Wylie Eminent Visiting Fellowship 2011 at the University of Queensland, Brisbane, Australia for a period of four weeks.

Federal Grants Held: (Principal Investigator Only)

- 1980-1983 United States Air Force, "Mechanisms of Radiation-Induced Emesis in Dogs", \$76,847 total direct costs.
- 1982-1988 National Institute of Health, "Mechanisms of Desensitization at Central Synapses", \$464,786 total direct costs.
- 1984-1986 Defense Nuclear Agency, "Mechanisms of Radiation-Induced Emesis in Dogs", \$330,504 total direct costs.
- 1986-1996 National Institute of Health, "Mechanisms of Excitatory Amino Acids Actions and Toxicity", 1986-1989 \$231,848 total direct costs; 1990-1996 \$562,926 total direct costs.
- 1989-1993 National Institute of Health, "Mechanisms of Lead Neurotoxicity" \$373,576 total direct costs
- 1990-1995 National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs and PCDFs at a Waste Site", D.O. Carpenter, P.I. \$5,783,419 total direct costs.
- 1995-2001 Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. A Central/Eastern European Environ/Occup Training Program, D.O. Carpenter, P.I. \$657,520 total costs.
- 1995-2001 National Institute of Environmental Health Sciences, Superfund Basic Research Program, "Multidisciplinary Study of PCBs," D.O. Carpenter, P.I. \$12,653,709 total direct costs.
- 1998-1999 Environmental Protection Agency, A Indoor Air Risk at Akwesasne - Pilot Project, D.O. Carpenter, P.I. \$9,996 total costs.
- 2000-2002 Association Liaison Office for University Cooperation in Development, A Cooperative Program in Environmental Health between the Institute of Public Health at Makerere University, Kampala, Uganda and the School of Public Health, University at Albany, USA, D.O. Carpenter, P.I. \$96,432 total costs.
- 2001-2007 Fogarty International Center, National Institutes of Health, International Training Program in Environmental and Occupational Health. A Multidisciplinary Environmental Health Training, D.O. Carpenter, P.I. \$850,000 total costs.
- 2006-2011 Pakistan-US Science and Technology Cooperative Program (US National Academy of Sciences). "Association of particulate matter with daily morbidity in an urban population," D.O. Carpenter, P.I., \$391,104 total costs.
- 2009-2013 Exploratory Center on Minority Health and Health Disparities in Smaller Cities. Project 2: Environmental contaminants and reproductive health of Akwesasne Mohawk women.

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\$387,825 for year 1. D.O. Carpenter, Co-PI.

- 2010-2013 Department of the Army, "Gulf War Illness: Evaluation of an Innovative Detoxification Program: D.O. Carpenter, P.I., \$636,958 total costs.
- 2010-2013 Higher Education for Development of the United States Agency for International Development, "Drinking Water Supply, Sanitation, and Hygiene Promotion : Health Interventions in Two Urban Communities of Kampala City and Mukono Municipality, Uganda". D. O. Carpenter, P.I., \$299,736 total costs.
- 2011-2016 National Institute of Environmental Health Sciences (1R01ES019620), "Protecting the health of future generations: Assessing and preventing exposures." PK Miller, FA von Hippel, CL Buck and DO Carpenter, Co-P.I.s, \$471,521 for the period 8/08/11-4/30/12, \$2,354,871 for the period 2011-2016.

Research Interests:

- Exposure to persistent organic pollutants and risk of diabetes, cardiovascular disease, and hypertension.
- Cognitive and behavioral effects of environmental contaminants on children (IQ, ADHD) and older adults (dementias, Parkinson's Disease and ALS).
- Ionizing and non-ionizing radiation biology.
- Effects of air pollution on respiratory and cardiovascular function.

Other Professional Activities:

Host, The Public Radio Health Show (a 30 min public health information show carried on 170+ stations nationwide), plus the Armed Forces Radio Network and Voice of America, 1985-2001.

Authored a biweekly health column in The Troy Record, a local newspaper, 1997-1999.

Major Peer-Reviewed Publications:

1. Carpenter, D.O., Lundberg, A. and Norrsell, U. Effects from the pyramidal tract on primary afferents and on spinal reflex actions to primary afferents. Experientia, 18:337, 1962.
2. Carpenter, D.O., Engberg, I. and Lundberg, A. Presynaptic inhibition in the lumbar cord evoked from the brain stem. Experientia, 18:450, 1962.
3. Carpenter, D.O., Lundberg, A. and Norrsell, U. Primary afferent depolarization evoked from the sensorimotor cortex. Acta Physiol. Scand., 59:126-142.
4. Carpenter, D.O., Engberg, I., Funkenstein, H. and Lundberg, A. Decerebrate control of reflexes to primary afferents. Acta Physiol. Scand., 59:424-437, 1963.
5. Carpenter, D.O., Engberg, I. and Lundberg, A. Differential supraspinal control of inhibitory and excitatory actions from the FRA to ascending spinal pathways. Acta Physiol. Scand., 63:103-110, 1965.
6. Henneman, E., Somjen, G.G. and Carpenter, D.O. Excitability and inhibibility of motoneurons of different sizes. J. Neurophysiol., 28:599-620, 1965.

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7. Henneman, E., Somjen, G.G. and Carpenter, D.O. Functional significance of cell size in spinal motoneurons. J. Neurophysiol., 28:560-580, 1965.
8. Somjen, G.G., Carpenter, D.O. and Henneman, E. Selective depression of alpha motoneurons of small size by ether. J. Pharmacol., 148:380-385, 1965.
9. Somjen, G., Carpenter, D.O. and Henneman, E. Response of motoneurons of different sizes to graded stimulation of supraspinal centers of the brain. J. Neurophysiol., 28:958-965, 1965.
10. Carpenter, D.O., Engberg, I. and Lundberg, A. Primary afferent depolarization evoked from the brain stem and the cerebellum. Arch. Ital. Biol., 104:73-85, 1966.
11. Carpenter, D.O. and Henneman, E. A relation between the threshold of stretch receptors in skeletal muscle and the diameter of axons. J. Neurophysiol., 29:353-368, 1966.
12. Carpenter, D.O. Temperature effects on pacemaker generation, membrane potential, and critical firing threshold in Aplysia neurons. J. Gen. Physiol., 50:1469-1484, 1967.
13. Chase, T.N., Breese, G., Carpenter, D., Schanberg, S. and Kopin, I. Stimulation-induced release of serotonin from nerve tissue. Adv. Pharmacol., 6A:351-364, 1968.
14. Carpenter, D.O. and Alving, B.O. A contribution of an electrogenic Na^+ pump to membrane potential in Aplysia neurons. J. Gen. Physiol., 52:1-21, 1968.
15. Olson, C.B., Carpenter, D.O. and Henneman, E. Orderly recruitment of muscle action potentials. Arch. Neurol., 19:591-597, 1968.
16. Carpenter, D.O. Membrane potential produced directly by the Na^+ pump in Aplysia neurons. Comp. Biochem. Physiol., 35:371-385, 1970.
17. Carpenter, D.O. and Gunn, R. The dependence of pacemaker discharge of Aplysia neurons upon Na^+ and Ca^{++} . J. Cell. Physiol., 75:121-127, 1970.
18. Kraus, K.R., Carpenter, D.O. and Kopin, I. R. Acetylcholine-induced release of norepinephrine in the presence of tetrodotoxin. J. Pharmacol. Exp. Therap., 73:416-421, 1970.
19. Barker, J.L. and Carpenter, D.O. Thermosensitivity of neurons in the sensorimotor cortex of the cat. Science, 169:597-598, 1970.
20. Carpenter, D.O., Hovey, M.M. and Bak, A. Intracellular conductance of Aplysia neurons and squid axon as determined by a new technique. Intl. J. Neurosci., 2:35-48, 1971.
21. Carpenter, D.O., Breese, G., Schanberg, S. and Kopin, I. Serotonin and dopamine: Distribution and accumulation in Aplysia nervous and non-nervous tissues. Int. J. Neurosci., 2:49-56, 1971.
22. Hovey, M.M., Bak, A.F. and Carpenter, D.O. Low internal conductivity of Aplysia neuron somata. Science, 176:1329-1331, 1972.
23. Carpenter, D.O. Electrogenic sodium pump and high specific resistance in nerve cell bodies of the squid. Science, 179:1336-1338, 1973.
24. Carpenter, D.O. and Rudomin, P. The organization of primary afferent depolarization in the isolated spinal cord of the frog. J. Physiol. (Lond.), 229:471-493, 1973.
25. Shain, W., Green, L.A., Carpenter, D.O., Sytkowski, A.J. and Vogel, Z. Aplysia acetylcholine receptors: Blockage by and binding of α -bungarotoxin. Brain Res., 72:225-240, 1974.
26. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Mammalian cold receptor afferents: Role of an electrogenic sodium pump in sensory transduction. Brain Res., 73:156-160, 1974.
27. Saavedra, J.M., Brownstein, M.J., Carpenter, D.O. and Axelrod, J. Octopamine: Presence in single neurons in Aplysia suggests neurotransmitter function. Science, 185:364-365, 1974.

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28. Willis, J.A., Gaubatz, G.L. and Carpenter, D.O. The role of the electrogenic sodium pump in modulation of pacemaker discharge of Aplysia neurons. J. Cell. Physiol., 84:463-472, 1974.
29. Brownstein, M.J., Saavedra, J.M., Axelrod, J., Zeman, G.H. and Carpenter, D.O. Coexistence of several putative neurotransmitters in single identified neurons of Aplysia. Proc. Natl. Acad. Sci. (USA), 71:4662-4665, 1975.
30. Carpenter, D.O. and Gaubatz, G.L. Octopamine receptors on Aplysia neurons mediate hyperpolarization by increasing membrane conductance. Nature, 252:483-485, 1974.
31. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Afferent nerve fiber activity responding to temperature changes of the scrotal skin of the rat. J. Neurobiol., 38:601-612, 1975.
32. Carpenter, D.O. and Gaubatz, G.L. H₁ and H₂ histamine receptors on Aplysia neurons. Nature, 254:343-344, 1975.
33. Carpenter, D.O., Hovey, M.M. and Bak, A.F. Resistivity of axoplasm. II. Internal resistivity of giant axons of squid and Myxicola. J. Gen. Physiol., 66:139-148, 1975.
34. Zeman, G.H. and Carpenter, D.O. Asymmetric distribution of aspartate in ganglia and single neurons of Aplysia. Comp. Biochem. Physiol., 52C:23-26, 1975.
35. Pierau, Fr.-K., Torrey, P. and Carpenter, D.O. Effect of ouabain and potassium-free solution on mammalian thermosensitive afferents in vitro. Pflugers Arch., 359:349-356, 1975.
36. Swann, J.W. and Carpenter, D.O. The organization of receptors for neurotransmitters on Aplysia neurons. Nature, 258:751-754, 1975.
37. Yarowsky, P.J. and Carpenter, D.O. Aspartate: distinct receptors on Aplysia neurons. Science, 192:806-809, 1976.
38. Foster, K.R., Bidinger, J.M. and Carpenter, D.O. The electrical resistivity of aqueous cytoplasm. Biophys. J., 16:991-1001, 1976.
39. Carpenter, D.O., Greene, L.A., Shain, W. and Vogel, Z. Effects of eserine and neostigmine on the interaction of α -bungarotoxin with Aplysia acetylcholine receptors. Mol. Pharmacol., 12:999-1006, 1976.
40. Saavedra, J.M., Ribas, J., Swann, J. and Carpenter, D.O. Phenylethanolamine: A new putative neurotransmitter in Aplysia. Science, 195:1004-1006, 1977.
41. Carpenter, D.O., Swann, J.W. and Yarowsky, P.J. Effect of curare on responses to different putative neurotransmitters in Aplysia neurons. J. Neurobiol., 8:119-132, 1977.
42. Yarowsky, P.J. and Carpenter, D.O. GABA mediated excitatory responses on Aplysia neurons. Life Sci., 20:1441-1448, 1977.
43. Willis, J.A., Myers, P.R. and Carpenter, D.O. An ionophoretic module which controls electroosmosis. J. Electrophysiol. Tech., 6:34-41, 1977.
44. Yarowsky, P.J. and Carpenter, D.O. Receptors for gamma-aminobutyric acid (GABA) on Aplysia neurons. Brain Res., 144:75-94, 1978.
45. Carpenter, D.O., Gaubatz, G., Willis, J.A. and Severance, R. Effects of irradiation of Aplysia pacemaker neurons with 20 MeV electrons. Rad. Res., 76:32-47, 1978.
46. Yarowsky, P.J. and Carpenter, D.O. A comparison of similar ionic responses to gamma-aminobutyric acid and acetylcholine. J. Neurophysiol., 41:531-541, 1978.
47. Blum, B., Aufer, C.R. and Carpenter, D.O. A head holder and stereotaxic device for the rattlesnake. Brain Res. Bull., 3:271-274, 1978.

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48. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Dopamine-induced muscle contractions and modulation of neuromuscular transmission in Aplysia. Brain Res., 157:167-172, 1978.
49. Swann, J.W., Sinback, C.N. and Carpenter, D.O. Evidence for identified dopamine motor neurons to the gill of Aplysia. Neurosci. Lett., 10:275-280, 1978.
50. Keabian, P.R., Keabian, J.W. and Carpenter, D.O. Regulation of cyclic AMP in heart and gill of Aplysia by the putative neurotransmitters, dopamine and serotonin. Life Sci., 24:1757-1764, 1979.
51. Carpenter, D.O. Interchangeable association of neurotransmitter receptors with several ionophores. Brain Res. Bull., 4:149-152, 1979.
52. Pellmar, T.C. and Carpenter, D.O. Voltage-dependent calcium current induced by serotonin. Nature, 277:483-484, 1979.
53. Ruben, P.C., Swann, J.W. and Carpenter, D.O. Neurotransmitter receptors on gill muscle fibers and the gill peripheral nerve plexus in Aplysia. Canad. J. Physiol. Pharmacol., 57:1088-1097, 1979.
54. Pellmar, T.C. and Carpenter, D.O. Serotonin induces a voltage-sensitive calcium current in neurons of Aplysia californica. J. Neurophysiol., 44:423-439, 1980.
55. Parver, L.M., Auker, C. and Carpenter, D.O. Choroidal blood flow as a heat dissipating mechanism in the macula. Am. J. Ophthalmol., 89:641-646, 1980.
56. Mell, L.D., Jr. and Carpenter, D.O. Fluorometric determination of octopamine in tissue homogenates by high-performance liquid chromatography. Neurochem. Res., 5:1089-1096, 1980.
57. Braitman, D.J., Auker, C.R. and Carpenter, D.O. Thyrotropin-releasing hormone has multiple actions in cortex. Brain Res., 194:244-248, 1980.
58. Meszler, R.M., Auker, C.R. and Carpenter, D.O. Fine structure and organization of the infrared receptor relay, the lateral descending nucleus of the trigeminal nerve in pit vipers. J. Comp. Neurol., 196:571-584, 1981.
59. Auker, C.R., Parver, L.M., Doyle, T. and Carpenter, D.O. Choroidal blood flow: I. Ocular tissue temperature as a measure of flow. Arch. Ophthalmol., 100:1323-1326, 1982.
60. Parver, L.M., Auker, C., Carpenter, D.O. and Doyle, T. Choroidal blood flow: II. Reflexive control in the monkey. Arch. Ophthalmol., 100:1327-1330, 1982.
61. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Lateral olfactory tract transmitter: Glutamate, aspartate or neither? Cell. Mol. Neurobiol., 1:115-120, 1981.
62. Scappaticci, K.A., Dretchen, K.L., Carpenter, D.O. and Pellmar, T.C. Effects of furosemide on neural mechanisms in Aplysia. J. Neurobiol., 12:329-341, 1981.
63. Pellmar, T.C. and Carpenter, D.O. Cyclic AMP induces a voltage-dependent current in neurons of Aplysia californica. Neurosci. Lett., 22:151-157, 1981.
64. Parver, L., Auker, C. and Carpenter, D.O. Stabilization of macular temperature: The stabilizing effect of the choroidal circulation on the temperature environment of the macula. Retina, 2:117-120, 1982.
65. Green, R.W. and Carpenter, D.O. Biphasic responses to acetylcholine in mammalian reticulospinal neurons. Cell. Molec. Neurobiol., 1:401-405, 1981.
66. Hori, N., Auker, C.R., Braitman, D.J. and Carpenter, D.O. Pharmacologic sensitivity of amino acid responses and synaptic activation of in vitro prepyriform neurons. J. Neurophysiol., 48:1289-1301, 1982.

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67. Slater, N.T. and Carpenter, D.O. Blockade of acetylcholine-induced inward currents in *Aplysia* neurons by strychnine and desipramine: effect of membrane potential. Cell. Molec. Neurobiol., 2:53-58, 1982.
68. Swann, J.W., Sinback, C.N., Pierson, M.G. and Carpenter, D.O. Dopamine produces muscle contractions and modulates motoneuron-induced contractions in *Aplysia* gill. Cell. Molec. Neurobiol., 2:291-308, 1982.
69. Swann, J.W., Sinback, C.N., Keabian, P.R. and Carpenter, D.O. Motoneurons which may utilize dopamine as their neurotransmitter. Cell. Molec. Neurobiol., 2:309-324, 1982.
70. Auker, C.R., Meszler, R.M. and Carpenter, D.O. Apparent discrepancy between single unit activity and ¹⁴C-deoxyglucose labelling in the optic tectum of the rattlesnake. J. Neurophysiol., 49:1504-1516, 1983.
71. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Snyder, S.H. Vipoxin both activates and antagonizes three types of acetylcholine response in *Aplysia* neurons. Brain Res., 278:266-270, 1983.
72. French-Mullen, J.M.H., Hori, N., Nakanishi, H., Slater, N.T. and Carpenter, D.O. Asymmetric distribution of acetylcholine receptors and M channels on prepyriform neurons. Cell. Molec. Neurobiol., 3:163-182, 1983.
73. Carpenter, D.O., Briggs, D.B. and Strominger, N. Responses of neurons of canine area postrema to neurotransmitters and peptides. Cell. Molec. Neurobiol., 3:113-126, 1983.
74. Slater, N.T. and Carpenter, D.O. Blocking kinetics at excitatory acetylcholine responses on *Aplysia* neurons. Biophys. J., 45:24-25, 1984.
75. Chesnut, T.J. and Carpenter, D.O. Two-component desensitization of three types of responses to acetylcholine in *Aplysia*. Neurosci. Lett., 39:285-290, 1983.
76. Haas, H.L., Jeffreys, J.G.R., Slater, N.T. and Carpenter, D.O. Modulation of low calcium induced field bursts in the hippocampus by monoamines and cholinomimetics. Pflugers Arch., 400:28-33, 1984.
77. Parvar, L.M., Auker, C.R. and Carpenter, D.O. Choroidal blood flow. III. Reflexive control in human eyes. Arch. Ophthalmol., 101:1604-1606, 1983.
78. Slater, N.T., Haas, H.L. and Carpenter, D.O. Kinetics of acetylcholine-activated cation channel blockade by the calcium antagonist D-600 in *Aplysia* neurons. Cell. Molec. Neurobiol., 3:329-344, 1983.
79. McCreery, M.J. and Carpenter, D.O. Modulation of neuronal responses to L-glutamate in *Aplysia*. Cell. Molec. Neurobiol., 4:91-95, 1984.
80. Carpenter, D.O., Briggs, D.B. and Strominger, N. Peptide-induced emesis in dogs. Behav. Brain Res., 11:277-281, 1984.
81. French-Mullen, J.M.H., Hori, N. and Carpenter, D.O. N-methyl-D-aspartate and L-aspartate activate distinct receptors in prepyriform cortex. Cell. Molec. Neurobiol., 4:185-189, 1984.
82. Slater, N.T. and Carpenter, D.O. A study of the cholinolytic actions of strychnine using the technique of concentration jump relaxation analysis. Cell Molec Neurobiol 4:263-271, 1984.
83. Slater, N.T., Hall, A.F. and Carpenter, D.O. Kinetic properties of cholinergic desensitization in *Aplysia* neurons. Proc. Roy. Soc. Lond. B, 223:63-78, 1984.
84. Akaike, N., Hattori, K., Oomura, Y. and Carpenter, D.O. Bicuculline and picrotoxin block gamma-aminobutyric acid-gated Cl⁻ conductance by different mechanisms. Experientia, 41:70-71, 1985.

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85. Slater, N.T., Carpenter, D.O., Freedman, J.E. and Synder, S.H. Dual effects of the snake venom polypeptide vipoxin on receptors for acetylcholine and biogenic amines in *Aplysia* neurons. Neurosci., 14:723-733, 1985.
86. Mizuno, Y., Oomura, Y., Hori, N. and Carpenter, D.O. Action of vasopressin on CA1 pyramidal neurons in rat hippocampal slices. Brain Res., 309:241-246, 1984.
87. Slater, N.T., Hall, A.F. and Carpenter, D.O. Trifluoperazine and calcium antagonists accelerate cholinergic desensitization in *Aplysia* neurons. Brain Res., 329:275-279, 1985.
88. ffrench-Mullen, J.M.H., Koller, K., Zaczek, R., Coyle, J.T., Hori, N. and Carpenter, D.O. N-acetylaspartylglutamate: Possible role as the neurotransmitter of the lateral olfactory tract. Proc. Nat. Acad. Sci., 82:3897-3900, 1985.
89. Greene, R.W. and Carpenter, D.O. Actions of neurotransmitters on pontine medial reticular formation neurons of the cat. J. Neurophysiol., 54:520-531, 1985.
90. Hori, N., ffrench-Mullen, J.M.H. and Carpenter, D.O. Kainic acid responses and toxicity show pronounced Ca^{2+} dependence. Brain Res., 358:380-384, 1985.
91. Gaillard, W.D. and Carpenter, D.O. Spectra of neurotransmitter receptors and ionic responses on cerebral A and B neurons in *Aplysia californica*. Brain Res., 373:303-310, 1986.
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93. ffrench-Mullen, J.M.H., Hori, N. and Carpenter, D.O. A comparison on the effects of quinolinate and N-methyl-aspartate on neurons in rat piriform cortex. Neurosci. Lett., 63:66-70, 1986.
94. ffrench-Mullen, J.M.H., Hori, N. and Carpenter, D.O. Receptors for the excitatory amino acids on neurons in rat pyriform cortex. J. Neurophysiol., 55:1283-1294, 1986.
95. Slater, N.T., David, J.A. and Carpenter, D.O. Relaxation studies on the interaction of hexamethonium with acetylcholine-receptor channels in *Aplysia* neurons. Cell. Molec. Neurobiol., 6:191-211, 1986.
96. Leung, M.K., S.-Rozsa, K., Hall, A., Kuruvilla, S., Stefano, G.B. and Carpenter, D.O. Enkephalin-like substance in *Aplysia* nervous tissue and actions of leu-enkephalin on single neurons. Life Sci., 38:1529-34, 1986.
97. Slater, N.T., Filbert, M. and Carpenter, D.O. Multiple interactions of anticholinesterases with *Aplysia* acetylcholine responses. Brain Res., 375:407-412, 1986.
98. Carpenter, D.O. and Briggs, D.B. Insulin excites neurons of the area postrema and causes emesis. Neurosci. Lett., 68:85-89, 1986.
99. Carpenter, D.O., Briggs, D.B., Knox, A.P. and Strominger, N.L. Radiation-induced emesis in the dog: Effects of lesions and drugs. Rad. Res., 108:307-316, 1986.
100. Briggs, D.B. and Carpenter, D.O. Excitation of neurons in the canine area postrema by prostaglandins. Cell. Molec. Neurobiol., 6:421-426, 1986.
101. Chesnut, T.J., Carpenter, D.O. and Strichartz, G.R. Three effects of venom from *conus striatus* on the delayed rectifier potassium current of molluscan neurons. Toxicon, 25:267-278, 1987.
102. Yakushiji, T., Tokutomi, N., Akaike, N. and Carpenter, D.O. Agonists of GABA responses, studied using internally perfused frog dorsal root ganglion neurons. Neuroscience 22:1123-1133, 1987.
103. Akaike, N., Yakushiji, T., Tokutomi, N. and Carpenter, D.C. Multiple mechanisms of antagonism of GABA responses. Cell. Molec. Neurobiol., 7:97-103, 1987.

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104. Hori, N., Galeno, T. and Carpenter, D.O. Responses of pyriform cortex neurons to excitatory amino acids: Voltage dependence, conductance changes and effects of divalent cations. Cell. Molec. Neurobiol., 7:73-90, 1987.
105. Oyama, Y., King, W.M. and Carpenter, D.O. Edrophonium-induced membrane current in single neurons physically isolated from *Aplysia californica*. Brain Res., 438:95-100, 1988.
106. Jahan-Parwar, B., S.-Rozsa, K., Salanki, J., Evans, M.L. and Carpenter, D.O. *In vivo* labeling of serotonin containing neurons by 5,7-dihydroxytryptamine in *Aplysia*. Brain Res., 426:173-178, 1987.
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